The Effect of Food Deprivation on Nociception in Formalin Test and Plasma Levels of Noradrenaline and Corticosterone in Rats

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ABSTRACT

Introduction: The concentration of noradrenalin and corticosterone as the two nociception modulators change after fasting or stress situation. The aim of present study was to investigate the effect of food deprivation on formalin-induced nociceptive behaviours and plasma levels of noradrenalin and corticosterone in rats.

Methods: Food was withdrawn 12, 24 and 48 h prior to performing the formalin test, but water continued to be available ad libitum. The formalin solution (50 µL, 2%) was injected into plantar surface of hind paw. The nociception responses of the animals during the first phase (1-7 minutes), the inter-phase (8-14), the phase 2A (15-60) and the phase 2B (61-90) was separately evaluated. The plasma concentrations of noradrenalin and corticosterone were measured using specific ELISA and IRA kits, according to manufacturer's instructions.

Results: In contrast to the increasing of 48 h food deprived animals during phase 2, the nociceptive behaviours of 12 and 24 h groups decreased through the interphase, phase 2A and phase 2B. The injection of formalin in the normal male rats significantly decreased the plasma level of noradrenalin and corticosterone. Food deprivation for 12 and 24 h increased noradrenalin level significantly in comparison with control group which has caused by fasting induced antinociceptive behaviours. There was no significant change in food deprivation for 48 h group. Food deprivation for 12, 24 and 48 h had no effect on corticosterone level in male rats.

Discussion: The present study emphasizes that the acute food deprivation diminished the nociceptive behaviours in the formalin test and show a correlation with increase in plasma noradrenalin level.

1. Introduction

Both short-term and intermittent food deprivation are well known to have antinociceptive effect, which several neuromodulatory systems such as endogenous opioid system and adrenocortical hormones are known to be involved (Bodnar, Romero, & Kramer, 1988; Hamm & Knisely, 1986). Norepinephrine participates in descending pain inhibitory system. Brainstem nuclei A1–A7 such as locus coeruleus in centrally and sympathetic nerves in peripherally are the main sources of norepinephrine. Locus coeruleus in the pons has most projection to the dorsal horn of spinal cord (Proudfit, 1988; Kwiat & Basbaum, 1992) and has a key role in noradrenergic pain modulation. Locus coeruleus stimulation releases norepinephrine (Hentall, Mesigil, Pinzon, & Noga, 2003) and produces analgesia that is prevented by alpha-2-adrenoceptor antagonist's administration (Jones, 1991; Proudfit, 1988). Furthermore, norepinephrine is released by peripheral noxious stimulation (Takagi, Shiomi, Kuraishi, Fukui, & Ueda, 1979; Tyce &

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